* Trend test. # time to tPA and @ MI location used as covariates. Median time to ST resolution increased significantly as dose increased, a result opposite to that expected.
Conclusion: In this randomized, dose-ranging trial, rPSGL-Ig, given in combination with tPA to a maximum dose of 75 mg, did not significantly improve ST resolution, infarct size, ejection fraction or clinical outcomes.

11:30 a.m.

881FO-5 Concerted Action of Various Angiogenic Factors During Acute Myocardial Infarction in Patients: The Three Lines of Defense

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Background: It is well documented that angiogenesis plays a key role in the process of ventricular remodeling following acute myocardial infarction (AMI). However, the exact contribution of each of the numerous angiogenic agents in this process is still an issue of debate. We sought to evaluate the role of free Insulin-like Growth Factor-1 (fIGF-1), basic Fibroblast Growth Factor (bFGF) and Angiogenin (ANG), in AMI patients.
Methods: 40 pts with first attack of AMI admitted and thrombolysed during the acute phase, were examined for plasma levels of fIGF-1, bFGF and ANG, measured by ELISA, and compared to those of 20 normal controls (NC) with mean values: 7,78 ± 0,75 ng/ml for fIGF-1, 1,62 ± 0,53 pg/ml for bFGF and 210,12 ± 25,6 ng/ml for ANG. Plasma samples were collected on hospital admission (0 hours) and 3h, 6h, 9h, 12h, 18h, 24h, 36h, 48h, 3 days, 4d, 5d, 7d, 15d and 30d thereafter.
Results: Data is expressed as mean values ± SEM. For the statistical analysis the non-parametric Wilcoxon test was used with $p<0,05$ compared to NC (*) and the relevant plasma value on admission (^). fIGF-1 exhibited significantly high initial plasma levels at 0h (10,92 ± 0,93*) followed by a gradual increase up to a maximum at 36h (16,64 ± 2,12*^) succeeded by a progressive decline up to a minimum level at 30d (12,14 ± 1,35*). bFGF developed a significant early peak at 0h (5,27 ± 1,13*) followed by a steep decline leading to a nadir at 24h (1,63 ± 0,46^), that was reversed leading to a late peak at 15d (8,93 ± 2,06*). ANG also exhibited a double- peak increase at 0h (327,12 ± 31,75*) as well as at 4d (305,86 ± 29,44*) with the lowest plasma value at 24h (233,74 ± 18,32^).
Conclusions: Thus, the activation of the numerous angiogenic agents seems to be triggered at different time points. Some of them exhibit an immediate peak shortly after AMI onset, probably as a result of acute hypoxia, while others develop an intermediate or a late peak, therefore exerting their cardioprotective potential in three distinct lines of defense. At the same time, they all act in concert to stimulate angiogenesis either by playing a pivotal role in its induction, or by manipulating its multiple steps later in an attempt to modulate ventricular remodeling and preserve cardiac function.

11:45 a.m.

881FO-6 PIA Polymorphism and Myocardial Salvage in Patients With Acute Myocardial Infarction Treated With Stenting or Thrombolysis

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Background: PIA2 allele of glycoprotein IIIa gene has been shown to be associated with a higher risk for myocardial infarction and complications after coronary intervention. It is not clear, whether PIA2 allele carriage influences the degree of myocardial salvage in patients with acute myocardial infarction (AMI) treated with thrombolysis or stenting.
Methods: We analyzed 292 patients enrolled in STOPAMI1 and STOPAMI2 randomized trials that compared stenting plus abciximab with thrombolysis (alteplase alone or alteplase plus abciximab) in AMI. PIA genotype was performed with a TaqMan assay. Technetium-99m sestamibi was injected before and 1-2 weeks after reperfusion treatment to calculate the initial perfusion defect, final infarct size, and the proportion of initial defect salvaged with reperfusion (salvage index). Mortality was assessed in a period of 18 months.
Results: The genotype distribution was as follows: PIA2/A2 in 3.4%, PIA1/A2 in 24.7% and PIA1/A1 in 71.9% of patients. There were no significant differences between PIA2 allele carriers and PIA1/A1 patients in salvage index (0.45 vs 0.41, P=0.25), final infarct size (16.8% vs. 18.4% of left ventricle, P=0.46) as well as 18-month mortality (8.5% vs.7.1%, P=0.69). The relation between PIA2 allele presence and salvage index was absent both in stenting and thrombolysis group.
Conclusions: PIA2 allele has no influence on the degree of myocardial salvage achieved with stenting or thrombolysis in patients with AMI.